EXHIBIT 26

ORIGINAL ARTICLE

Head injuries and Parkinson's disease in a case-control study

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ABSTRACT

Background Head injury is a hypothesised risk factor for Parkinson's disease, but there is a knowledge gap concerning the potential effect of injury circumstances (eg, work-related injuries) on risk. The objective of this study is to address this gap while addressing issues of recall bias and potential for reverse causation by prediagnosis symptoms.

Methods We conducted a population based case-control study of Parkinson's disease in British Columbia, Canada (403 cases, 405 controls). Interviews queried injury history; whether injuries occurred at work, in a motor vehicle accident or during sports. Participants were also asked to report their suspicions about the causes of Parkinson's disease to provide an indicator of potential recall bias. Associations were estimated with logistic regression, adjusted for age, sex and smoking history.

Results Associations were strongest for injuries involving concussion (OR: 2.08, 95% CI 1.30 to 3.33) and unconsciousness (OR: 2.64, 95% CI 1.39 to 5.03). Effects remained for injuries that occurred long before diagnosis and after adjustment for suspicion of head injury as a cause of Parkinson's disease. Injuries that occurred at work were consistently associated with stronger ORs, although small numbers meant that estimates were not statistically significant.

Conclusions This study adds to the body of literature suggesting a link between head injury and Parkinson's disease and indicates further scrutiny of workplace incurred head injuries is warranted.

What is already known on this subject

- Several previous studies have found a link between Parkinson's disease and head injuries, although this result is not always observed and has been attributed to reverse causation due to early symptoms.
- Recall bias has been suspected to contribute to risk relationships of self-reported exposures and Parkinson's disease.

What this paper adds

- This study is the first (to our knowledge) that compares risks of head injuries incurred at work to those incurred in other circumstances.
- Injuries resulting in concussion and loss of consciousness were most strongly associated with Parkinson's disease.
- Of all injury circumstances, associations of occupational head injuries with Parkinson's disease were strongest, though this was not statistically significant.
- Adjusting for participants' suspicions about the role of head injuries in causing Parkinson's disease did not substantially attenuate associations.

INTRODUCTION

Head injuries are hypothesised to affect risk for neurodegenerative diseases such as Parkinson's disease (PD). While there is possible mechanistic support for this hypothesis[1] and analogy may be drawn to parkinsonian symptoms and movement disorders arising after brain injury,[2] epidemiological findings are inconsistent. Several epidemiological studies have found a relationship between prior head injury and idiopathic PD,[3–7] while others have not.[8 9] Rugbjerg *et al*[10] reported an association between hospitalisation for head injury and PD, but found that the association was largely explained by injuries occurring shortly before PD diagnosis, suggesting possible reverse causation. However, Goldman *et al*[7] found an effect of head injury on PD risk in twins even when restricting to injuries occurring 10 or more years prior to diagnosis. Such findings emphasise the importance of considering injury timing in relation to PD diagnosis, particularly due to the insidious onset of PD.

The nature of previous head injuries appears to be important to risk. Severe injuries[5] and those entailing loss of consciousness[3 5] seem more strongly associated with PD. Few studies have considered injury circumstances in analyses of risk. We were interested in PD and head injuries that occurred at work, but could find no previous studies of this issue. Although data are limited, work-related injuries could represent a more severe subset of all head injuries. Rickels *et al*[11] found that 15% of all head injuries in Germany occurred at or while travelling for work. Masson *et al*[12] reported that of severe brain injuries presenting at emergency departments in Aquitaine, France, 32% occurred at work.

Using population-based lists, we recruited a case-control study sample in British Columbia, Canada, and estimated the association between PD and head injury, considering the type of injury, the circumstances in which the injury occurred (at work, in a motor vehicle accident or during sports), and the

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temporal separation from diagnosis (to address the possibility of reverse causation by prediagnosis symptoms). We also sought to address the challenge of recall bias, in which cases report more exposures of interest than controls due to greater knowledge of suspected disease causes and more careful scrutiny of their past exposures. In our study of pesticide exposure and PD, adjusting for participants' suspicions about the causes of PD reduced risk estimates.[13] Here we take a similar approach: adjusting estimates of association for suspicion of head injury as a cause of PD.

MATERIALS AND METHODS

The study population included residents of the greater Vancouver region and of Vancouver Island outside Victoria, between the ages of 40 years and 69 years, registered with the universal provincial health insurer (British Columbia Medical Services Plan or MSP), living in the community (not in long-term care facilities) and with sufficient English facility to complete the interview. The upper age limit was applied to maximise recall of occupational exposures. Potential controls were drawn from the MSP register, frequency matched to the potential case sample on age, sex and geographical location.

Due to the long survival in patients with PD,[14] this study included prevalent cases. Potential cases were those who were reimbursed by the provincial health insurer for antiparksonian drugs: levodopa; bromocriptine mesylate; pergolide mesylate; levodopa/benserazide hydrochloride; levodopa/carbidopa; or seligiline hydrochloride between 1995 and 2002. Using anti-PD drugs is an effective way to identify PD cases with high sensitivity,[15 16] although specificity may be reduced by off-label use of antiparkinsonian medications.[16] As a privacy protection technique, the Ministry of Health added a number of individuals to the potential case list who did not meet the drug reimbursement criteria. This sample was referred to as a 'camouflage', designed to mask the commonality between list members.

The British Columbia Ministry of Health required a two-stage contact procedure. First, clerical staff contacted potential participants with a letter, followed by telephone calls to request permission to pass contact information to the University of British Columbia study team. If permission was granted, the study team initiated a second round of contact to screen for eligibility and request participation in an in person interview. Study staff screened potential participants over the telephone: potential participants were asked about chronic conditions and medication use. Controls reporting PD in telephone screening were excluded (one person), as were candidate cases who reported not having PD and use of antiparkinsonian medications for other purposes. Our recruitment process has also been described elsewhere.[13 17]

Interviews were conducted using a detailed questionnaire covering complete occupational history (all jobs held and daily duties), medical history and personal habits. All interviews were conducted between 2001 and 2008. Interviews included a checklist of symptoms to verify case status. Each checklist was reviewed by a neurologist (JKCT). The following clinical diagnostic criteria for PD were used: (1) two of the following symptoms present on examination: parkinsonian tremor, rigidity, bradykinesia, masked facies, micrographia or postural imbalance; (2) absence of specific signs of other diseases that would account for these findings. Dates of PD diagnosis, first symptoms and first treatment were also recorded.

In the medical history section of the questionnaire, participants were asked to report any injury event for which they visited a physician. They were asked to report:

- the part(s) of the body injured
- the date of the injury
- whether the injury event took place at work, in a motor vehicle accident, during a hobby, or during sports. Participants could nominate more than one of these circumstances if applicable (eg, sports injury that occurred at work). Reports of injuries occurring in multiple categories of circumstances were rare (three total) and these persons were counted as exposed in each category nominated.
- a brief description of the injury (free form description)

These data were then used by an investigator (KT), blinded to case or control status, to identify all injuries involving the face or head. With assistance from the team neurologist (JKCT), these were classified in categories of increasing severity: those involving stitches to the face or head; those involving a concussion; and those involving unconsciousness (all unconsciousness events were also included in the concussion category).

To assess the potential for recall bias to influence the reporting of exposures, we asked an open-ended question near the end of the interview: 'What do you think causes PD?' Responses were categorised, blind to case or control status, to create a dichotomous variable indicating suspicion of head injury as a cause of PD.

Odds of PD in those with and without head injuries were compared using unconditional logistic regression. Distinct models were constructed for head injuries entailing stitches to the face or head, concussion and unconsciousness. Within each head injury type, separate models were created for:

1. Timing of injuries prior to diagnosis: These analyses assessed associations with PD when head injury exposure reports were censored at 5, 10 and 20 years prior to diagnosis to account for possible latency between injuries and PD and for the insidious onset of PD and possible premorbid symptoms. For all analyses, control subjects were randomly assigned a 'diagnosis date' from a case in their age and sex stratum to ensure risk exposure periods were comparable between cases and controls. Nine controls and 23 cases reported multiple head injury incidents. Although these were too few to examine the effects of each additional head injury, these participants were included in latency analyses using the date of the first head injury.
2. Effect of causal knowledge: These analyses used the dichotomous variable indicating suspicion of a causal link between head injury and PD as an adjustment variable in analyses. If this causal suspicion were positively related to injury reports and PD, consistent with recall bias, we expected attenuation of the main effect of head injury after adjustment.
3. Injury circumstances: Associations with PD were assessed for injuries that occurred at work, in a motor vehicle accident or during sports. Separate regression analyses were performed for each class of injury circumstances, with only those without any head injury included in the reference group.

Statistical significance of ORs was assessed with 95% CIs. All models were adjusted for sex, year of birth (in 5 year categories) and smoking (in pack-years).

RESULTS

Eight hundred and eight participants were recruited and interviewed (403 PD cases and 405 controls), from 2261 potential cases and 1522 potential controls identified by the British Columbia Ministry of Health based on the population level administrative lists described above. Please see figure 1 for a flow chart of the recruitment process and participation. Particularly notable were the large numbers of potential cases who were not eligible to participate in the study, including those who appeared to be the camouflage sample and the substantial

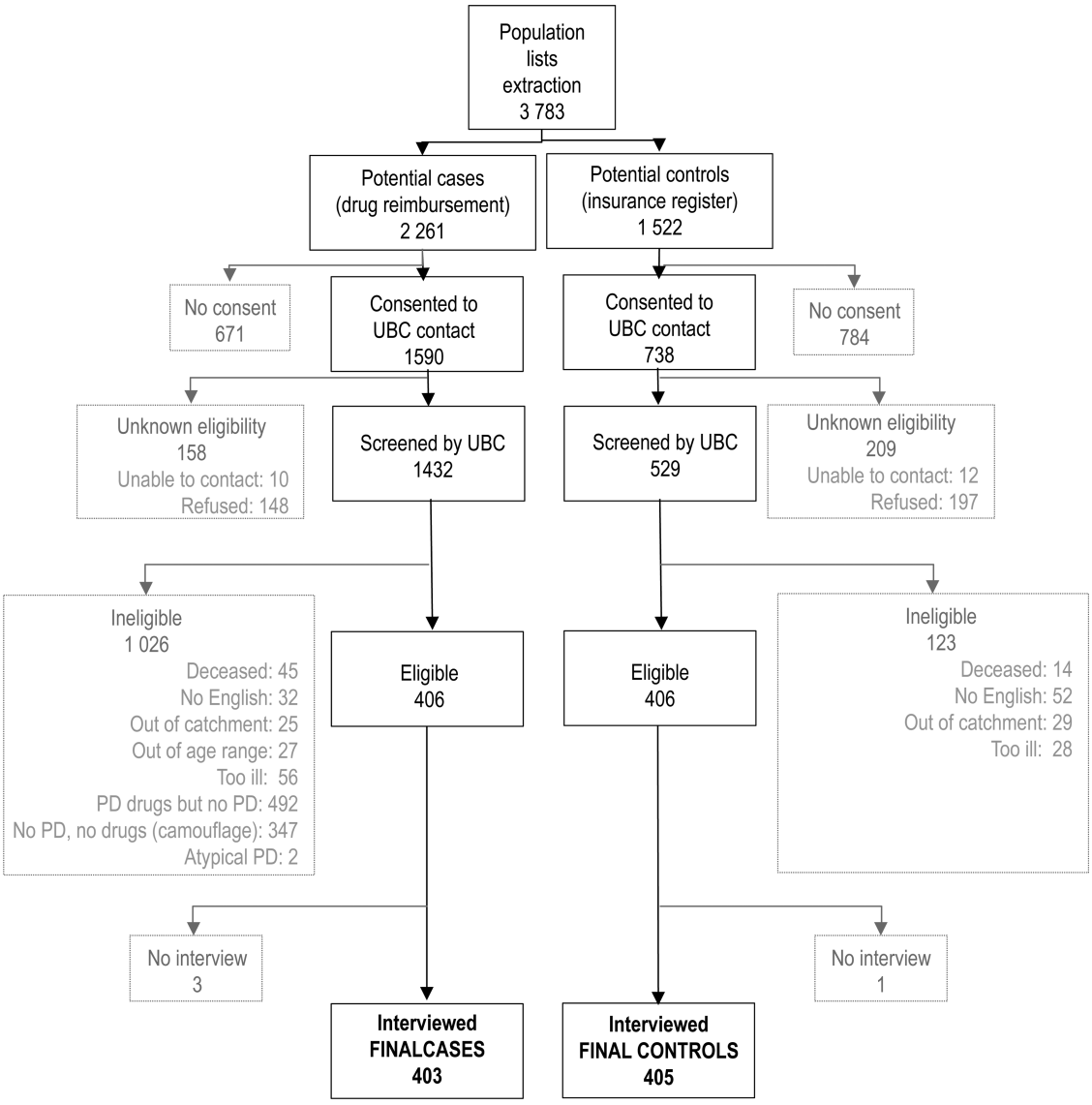

Figure 1 Recruitment and participation report for a case-control study of Parkinson's disease.

number of people using PD drugs for conditions other than PD. We calculated participation rates by correcting the denominator for the large number of potential participants who were not eligible to participate. Of those screened for eligibility, 77% of potential controls were eligible but only 28% of potential cases were eligible. If we assume that these proportions eligible would also be observed in those initially extracted had we been able ascertain their eligibility, we can assume that our eligible samples would consist of 633 cases (2261×0.28) and 1172 controls (1522×0.77). From this, we calculate a participation rate of 63.7% (403/633) for cases and 34.6% (405/1172) for controls. Table 1 summarises basic characteristics of the recruited sample.

One hundred and forty-three participants reported at least one previous head injury. Of these, 22 reported an injury at work, 49 in a motor vehicle accident, 27 during sports, 11 during a hobby and 39 in other circumstances. As there were few injuries occurring in hobbies other than sports, we did not examine associations for these circumstances. Overall, head injuries resulting in stitches to the face or head were not significantly associated with PD, although there were non-significant elevated risks for such injuries (table 2). There were consistent and moderately strong associations between PD and head injuries entailing concussion and loss of consciousness (table 2). These associations were not substantially attenuated by

Table 1 Characteristics of 403 cases with Parkinson's disease (PD) and 405 population-based controls in British Columbia, Canada

Characteristic	Cases N (%)	Controls N (%)
Men	266 (66.0)	204 (50.4)
Women	137 (34.0)	201 (49.6)
Birth year		
1929–1938	245 (60.8)	175 (43.2)
1939–1948	131 (32.5)	129 (31.9)
1949–1958	27 (6.7)	101 (25.0)
Indicated suspicion of head injury as a cause of PD	41 (10.2)	22 (5.4)
	Mean (SD)	Mean (SD)
Cumulative pack-years of smoking	11.4 (20.4)	15.4 (22.4)
Age (years) at diagnosis of Parkinson disease	56.0 (7.1)	–
Age (years) at the time of interview	65.0 (6.6)	62.2 (9.0)

Table 2 Results of logistic regression analyses estimating the association between Parkinson's disease and head injuries. Measures of associations in bold indicate statistical significance at p<0.05

	Cases/controls exposed	Cases/controls No head injury	OR (95% CI)
Stitches to face or head			
All injuries requiring stitches to face or head	30/24	311/354	1.31 (0.72 to 2.36)
>5 years prior to diagnosis	27/23	320/357	1.16 (0.63 to 2.12)
>10 years prior to diagnosis	26/21	323/360	1.26 (0.67 to 2.34)
>20 years prior to diagnosis	24/17	331/370	1.49 (0.76 to 2.90)
Adjusted for head injury causal knowledge	30/24	311/354	1.29 (0.71 to 2.32)
Injury circumstances:			
At work	7/3	311/354	2.07 (0.51 to 8.32)
In motor-vehicle crash	3/7	311/354	0.54 (0.13 to 2.23)
During sports	8/4	311/354	1.78 (0.51 to 6.23)
Concussion head injuries (includes unconsciousness)			
All injuries resulting in concussion	65/35	311/354	**2.08 (1.30 to 3.33)**
>5 years prior to diagnosis	59/30	320/357	**2.17 (1.32 to 3.55)**
>10 years prior to diagnosis	57/29	323/360	**2.08 (1.27 to 3.43)**
>20 years prior to diagnosis	50/23	331/370	**2.23 (1.30 to 3.82)**
Adjusted for head injury causal knowledge	65/35	311/354	**1.93 (1.20 to 3.10)**
Injury circumstances:			
At work	10/5	311/354	2.57 (0.80 to 8.23)
In motor-vehicle crash	27/16	311/354	1.88 (0.96 to 3.70)
During sports	9/8	311/354	1.59 (0.54 to 4.66)
Unconsciousness head injuries			
All injuries resulting in unconsciousness	39/15	311/354	**2.64 (1.39 to 5.03)**
>5 years prior to diagnosis	35/12	320/357	**2.94 (1.46 to 5.92)**
>10 years prior to diagnosis	35/12	323/360	**2.92 (1.45 to 5.87)**
>20 years prior to diagnosis	30/11	331/370	**2.76 (1.32 to 5.78)**
Adjusted for head injury causal knowledge	39/15	311/354	**2.46 (1.29 to 4.73)**
Injury circumstances:			
At work	8/3	311/354	3.28 (0.80 to 13.42)
In motor-vehicle crash	18/8	311/354	2.13 (0.89 to 5.11)
During sports	6/3	311/354	2.18 (0.49 to 9.65)

All analyses adjusted for age, sex and smoking.

censoring of head injury exposures at 5, 10 and 20 years prior to diagnosis. Table 3 lists the average interval between head injury and PD diagnosis among cases for each head injury type. Sixty-three (7.8%) participants suspected head injury as a cause of PD, and this causal suspicion was more common in cases (table 1) and those reporting a previous head injury, with 25 of the 143 participants reporting previous head injury nominating injury as a cause of PD and 38 of the 665 reporting no previous head injury citing injury as a suspected cause. However, associations between PD and concussion and loss of consciousness injuries remained moderately strong after adjustment for suspicion of head injury as a cause of PD (table 2).

Effect estimates for injuries incurred at work were consistently stronger than those noted for injuries resulting from motor vehicle accidents or during sports (table 2), although the small numbers of observed events meant that these associations were not statistically significant.

DISCUSSION

This study adds to the literature showing associations between PD and head injury.[3–7] Like Dick *et al*[3] and Bower *et al*,[5] we found that head injury events resulting in unconsciousness were most strongly related to PD. Unlike Rugbjerg *et al*,[10] we did not find evidence of reverse causation as associations were not attenuated with increasing intervals between head injury and diagnosis. Many of the reported head injuries occurred long before diagnosis, with average intervals of 30 years or more.

To our knowledge, this study is the first to examine distinct risk relationships for different circumstances producing head injuries. These analyses were limited by small numbers of observations in categories of head injury circumstances, but did suggest a possible pattern of increased ORs for injuries occurred while at work that may warrant future studies. Workplace injuries may have higher ORs for PD because injuries that occur at work are more severe, are better recalled due to requirements for workplace incident investigation, or a combination of these as more severe occupational injuries appear to be better recalled.[18] It is also possible that workplace head injuries may

Table 3 Intervals between types of head injuries and Parkinson's disease diagnosis observed in a case control study

Injury type	Number of cases with complete date of injury data	Years between head injury and diagnosis			
		Mean	SD	Minimum	Maximum
Unconsciousness	38	36.4	11.3	1	57
Concussion	62	35.9	11.6	1	58
Stitches to the face or head	30	37.3	16.6	0	61

combine with other workplace exposures[13 17 19] suspected to increase risk of PD.

Consideration of the circumstances of injury and the nature of the injury (eg, loss of consciousness) may help derive and refine mechanistic hypotheses relating PD to head injury[1] and could add to future studies of genetic susceptibility and head injury.[8] Our study did not examine a history of multiple head injuries: we did not observe sufficient numbers of participants who had incurred repeated head injuries to examine dose-response relationships. While our study did not enrol any professional athlete participants, future work on repeated occupational head injury and PD may be highly relevant to recent interest in the long-term effects of repeated head injury in professional athletes.[20]

Participation rates in this study were distinctly lower than desired. As we have written[19] and commented[21] elsewhere, our study recruitment was negatively affected by restrictions intended to protect privacy (such as requiring multiple contact stages and the inclusion of a large sample of individuals on our case list who did not meet the case definition). While the resulting rates are disappointingly low and may affect the validity of the data,[22] we argue that our results remain informative in the absence of other population-based studies addressing the current research objectives, although we will await replication or falsification in future studies. Anonymous register-based studies not requiring participant contact may be a good way to achieve excellent 'participation' and test our findings. However, administrative registers may not include data on head injury exposures at work without supplemental interview or contact with participants, which is increasingly restricted.[21 22]

Cases and controls were not well matched on age and sex, in part because the use of PD drugs for indications other than PD (in particular restless legs syndrome) was related to younger ages and female sex. Since the true case sample was older and more male than the potential case sample, frequency matching to the potential case extraction introduced differences. We used statistical adjustment to address sex frequency differences in cases and controls. To determine the efficacy of this adjustment, we conducted stratified analyses for head injury and several of our previously reported associations and found the trends to be similar to those with adjustment. One reason to feel confident about the ability of adjustment to address differences in age and sex is that these are variables that are likely to be reported with excellent accuracy and precision, with low probabilities of misclassification. Nonetheless, there is a possibility of residual confounding as 1 : 1 matching was not used.

This study relied on self-report. Two of the issues commonly limiting self-reports are non-differential recall error and recall bias. Non-differential recall errors occur because cases and controls may not remember or report some injury incidents; these tend to bias estimates of association towards the null. By contrast, recall bias occurs when cases are more likely to report exposures, which may bias estimates away from the null.[23]

We attempted to address recall bias in several ways. First, we used prompts on head injuries that required physician attention and their circumstances because prompted responses are less prone to recall bias than unprompted replies.[24] Second, we did not limit our queries to head injuries alone: we asked about all previous injuries affecting all parts of the body (partly to disguise our narrow interest in head injuries and partly to maximise the sensitivity of self-reports). Lastly we asked an open-ended question: 'What do you think causes PD?' and used this to create an adjustment variable indicating knowledge of the causal hypothesis. The fact that adjustment for this variable did not substantially affect our estimates of association suggests that recall bias alone is unlikely to explain the observed associations, although recall bias may not be fully addressed by these methods. This type of adjustment should be interpreted with caution, as there could be reasons other than recall bias why cases aware of a hypothesis could be more likely to report exposure and disease (eg, if patients become aware of suspected risk factors when researching their past exposures but the sensitivity of their reporting is not affected).

CONCLUSIONS

This case-control study provides further evidence that head injuries resulting in concussion or unconsciousness may increase risk of PD. It also suggests that occupational injuries should be examined further for potentially increased risk compared with other injury circumstances.

Contributors SAM, JKCT and KT designed the initial case-control study and obtained funding. MAH managed and cleaned data. All authors collaborated on designing the analytical plan. HS conducted statistical analyses. All authors had access to the study results and contributed to the interpretation of results. MAH drafted the manuscript with input from all authors.

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Competing interests None.

Patient consent Obtained.

Ethics approval University of British Columbia Behavioural Research Ethics Board.

Provenance and peer review Not commissioned; externally peer reviewed.

Data sharing statement The raw data for this study were collected after complex negotiations to protect participant privacy. The data cannot be released to external parties without permission from the British Columbia Ministry of Health and approval of the relevant research ethics boards.

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Notes